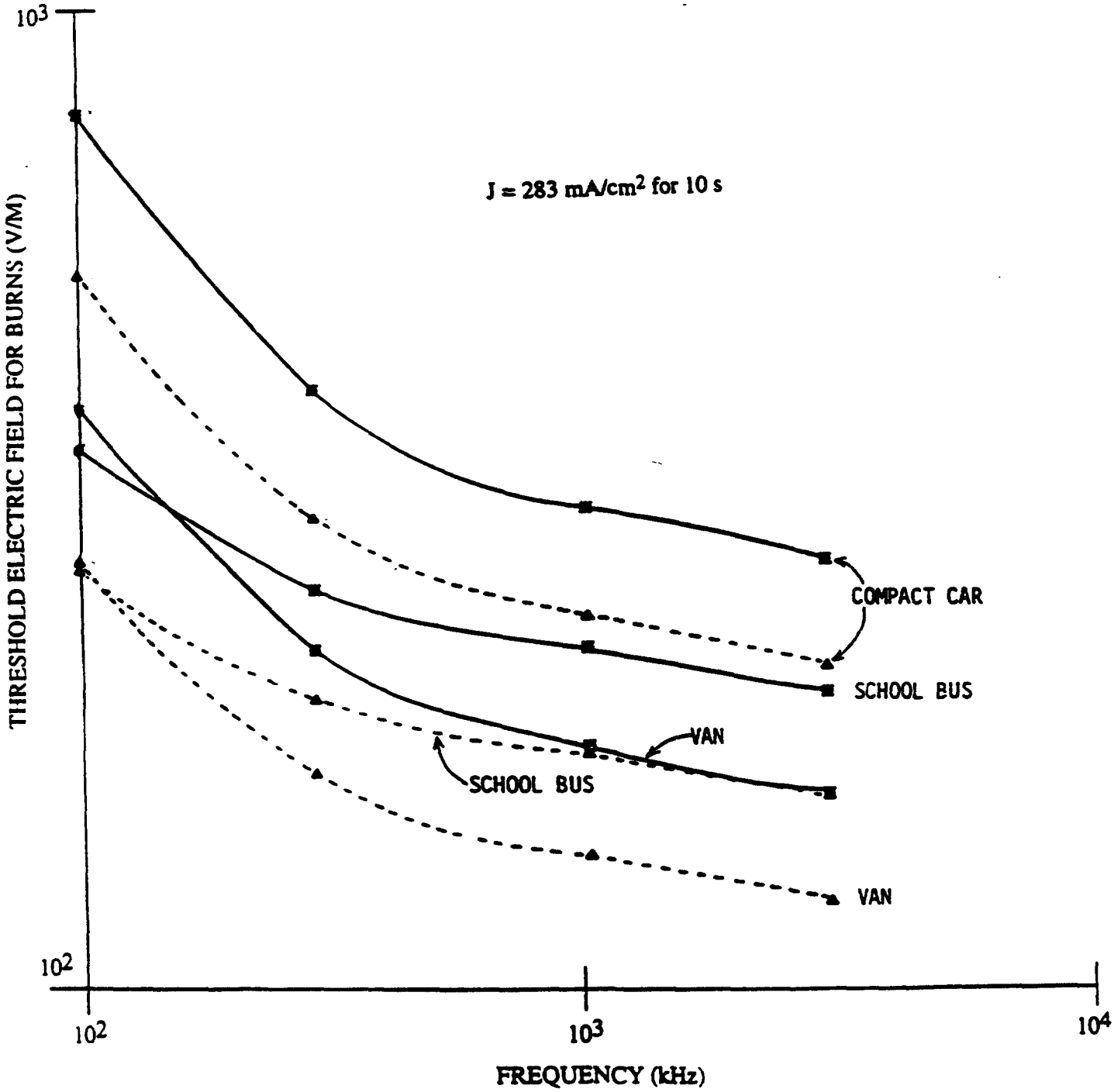

Fig. 3. Threshold E-field for producing burns in adult males (solid curves) and ten-year old children (dashed curves) in finger contact with various vehicles (contact area = 25 mm^2).

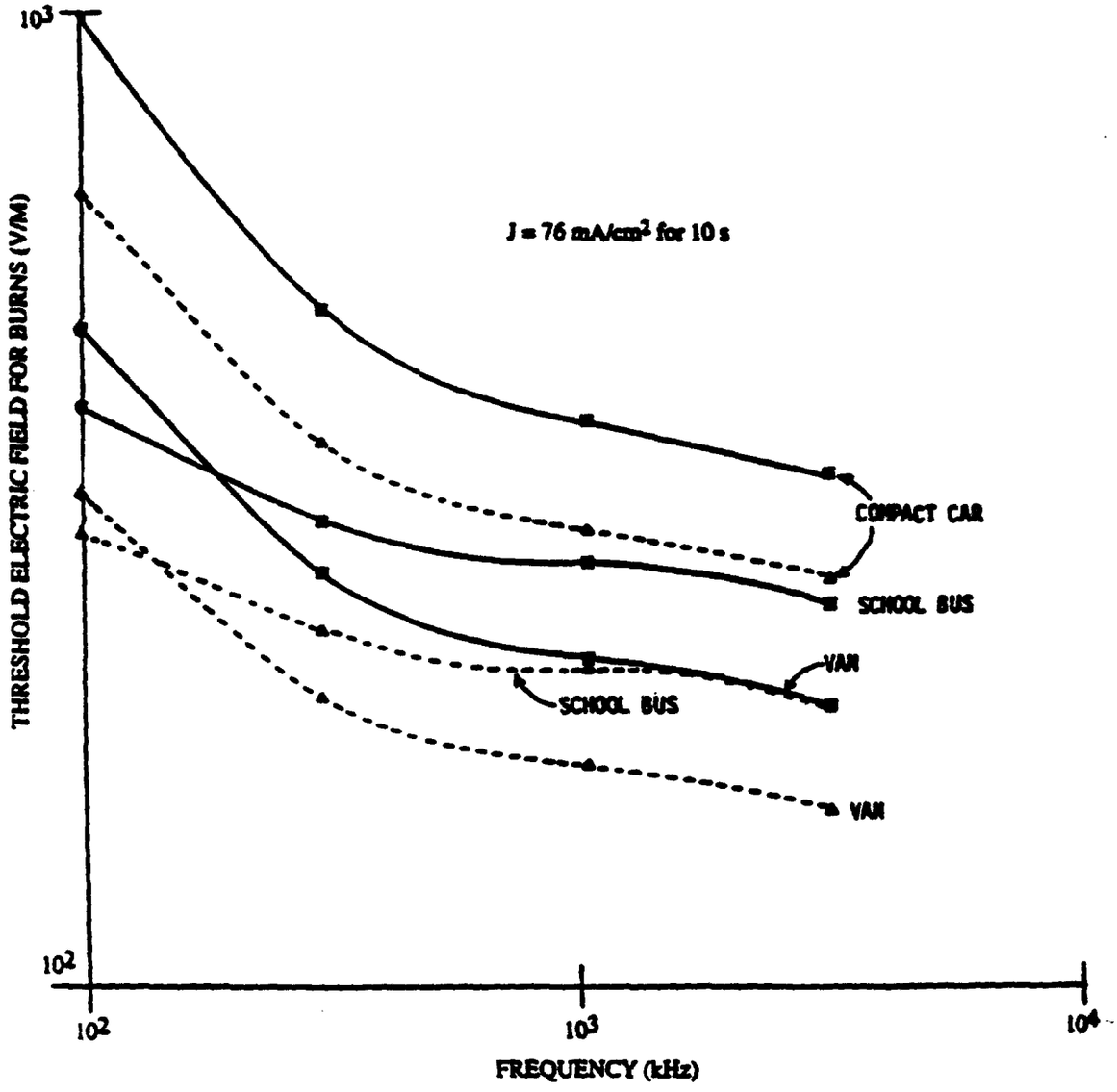

Fig. 4. Threshold E-field for producing burns in adult males (solid curves) and ten-year old children (dashed curves) in finger contact with various vehicles (contact area = 144 mm^2).

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NOTE TO READERS OF THE CRITERIA MONOGRAPHS

Every effort has been made to present information in the criteria monographs as accurately as possible without unduly delaying their publication. In the interest of all users of the environmental health criteria monographs, readers are kindly requested to communicate any errors that may have occurred to the Director of the International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland, in order that they may be included in corrigenda, which will appear in subsequent volumes.

frequencies (assessed as the decreased survival of implanted embryos and fetuses). Much experimental evidence suggests that acute or long-term RF exposures do not result in an increase in chromosome aberration frequency, when temperatures are maintained within physiological limits. One study reported an increased frequency of cytogenetic effects in mice exposed long-term at SARs between 0.05 and 20 W/kg. However, this study was not successfully corroborated using a different strain of mouse.

In general, the data in Table 28 suggest that the only exposures that are potentially mutagenic are those at high RF power densities, which result in substantial increase in temperature.

7.3.10 Cancer-related studies

A summary of cancer-related animal studies is given in Table 29. The number and types of studies are limited.

Exposure to RF levels sufficiently high to induce hyperthermia has generally resulted in tumour regression following transplantation of tumour cells (Preskorn et al., 1978; Roszkowski et al., 1980). In contrast, an increase in tumour progression has been observed in mice exposed long-term at lower, possibly thermogenic, SARs (Szmigielski et al., 1982). This effect was related to a non-specific stress. The authors suggested a transient shift in immune surveillance resulting in a lowering of resistance to neoplastic growth, as a likely explanation. Exposure at about 1 W/kg did not have any effect on melanoma growth in mice (Santini et al., 1988).

The effects of exposure on spontaneous or chemically-induced tumours have also been examined. In contrast to transplantation studies, these can test for an effect on the process of carcinogenesis. Two early studies (Prausnitz & Suskind, 1962; Skidmore & Baum, 1974), relevant to cancer induction, but in which the methodology was flawed in relation to an analysis of this end-point, are described for completeness. An increased incidence of monocytic leukosis (defined as a non-circulating neoplasm of white-blood cells) and lymphatic or myeloid leukaemia (defined as a circulating "leukosis") was reported in Swiss mice exposed to thermally significant levels (half the acute LD_{50}) of 9.27 GHz pulsed RF, for 5 days per week

Table 29. Cancer-related studies

Exposure conditions	Effect on exposed group	Reference
Transplanted tumour cells		
2.45 GHz (CW), 35 W/kg, for 20 min/day during days 11-14 of gestation; offspring injected with sarcoma cells at 16 days of age exposed for 36 days	Retarded tumour growth and tumour incidence in sarcoma-injected offspring of exposed pregnant mice; rectal temperature of dams rose over 2 °C; exposed mice had increased longevity	Preskorn et al. (1978)
2.45 GHz (CW), 25 W/kg, 2 h/day for 7 days; Injection of sarcoma cells in mice 14 days after, or just after, RF exposure	Temporary tumour regression followed by renewed tumour growth 12 days later, when exposure 14 days after tumour injection; accelerated tumour growth, if exposed before implantation of tumour; lung metastases increased	Roszkowski et al. (1980)
2.45 GHz (CW), 2-3 W/kg or 6-8 W/kg, 2 h/day, for 6 days/week; mice exposed from 6 weeks of age to 12 months of stress	RF caused increase in sarcoma colonies in lungs in mice injected intravenously with these cells; chronic via confinement caused similar increase in lung tumours as 2-3 W/kg, but 6-8 W/kg produced higher increase in tumours	Szmigielski et al. (1982)
2.45 GHz (CW and pulsed) 10 W/m^2, 1.2 W/kg prior to, and during, B16 melanoma tumour transplantation and growth; exposed for 2.5 h/day, 6 times/week for 15 days, prior to injection of melanoma cells, then exposed to same schedule until death	No difference in mean tumour surface area/animal, or in mean survival time between exposed or control mice	Santini et al. (1988)
Spontaneous or chemically-induced tumours		
2.45 GHz (CW), 2-3 W/kg or 6-8 W/kg, 2 h/day, for 6 days/week, mice exposed from 6 weeks of age to 12 months of stress	SAR-dependent acceleration of mammary tumours in mice genetically predisposed to these tumours, and acceleration of skin tumours in mice painted with the carcinogen 3,4-benzopyrene (BP)	Szmigielski et al. (1982)

Table 29 (continued)

Exposure conditions	Effect on exposed group	Reference
2.45 GHz (CW), 100 W/m^2 4-5 W/kg, for 2 h/day, 5-6 days/week for a few months	Increased development of chemically-induced hepatomas and sarcomas in mice; survival of exposed mice decreased; increased frequency of skin tumours in mice given subcarcinogenic dose of BP	Szmigielski et al. (1988)
2.45 GHz (10 μs pulses at 800 Hz) square wave-modulated at 8 Hz, 0.4 W/kg, continuous exposure at 2-27 months of age (lifetime study of rats)	Total incidence of neoplasia not significantly different from that in controls; however, increased number of primary malignancies (18) occurred early in exposed group compared with controls (5)	Guy et al. (1985)

for 59 days (Prausnitz & Susskind, 1962). However, the study suffered several deficiencies: leukosis and leukaemia were inadequately defined, infection may well have confounded the results, a large proportion of mice died without a cause of death being identified, and statistical analysis was absent (Roberts 1983; Kirk 1984).

Skidmore & Baum (1974) reported that exposure for 5 days per week for 33 weeks to very short pulses (5 ns rise time; 550 ms decay time) of high field strength (447 kV/m) pulsed at 5 Hz, resulted in a reduced incidence of leukaemia in AKR/J mice (which spontaneously develop a high incidence of lymphatic leukaemia between 26 and 52 weeks of age) compared with controls at the end of the exposure. However, the absence of a complete analysis of leukaemia incidence (and other causes of death) precludes any conclusion being drawn from this study. The authors also reported a zero incidence of mammary tumours in 1-year-old female Sprague-Dawley rats that had been exposed for 38 weeks; evaluation was probably premature for this end-point, the tumours occur spontaneously mainly in older rats. A later study (Baum et al., 1976) reported no effects on mammary tumour incidence and other lesions in rats exposed for 94 weeks.

Two studies merit particular attention. The long-term exposure of mice at SARs of between 2 and 8 W/kg resulted in an increase in

the number of sarcoma cell colonies in the lungs (following the injection of sarcoma cells), as shown in Fig. 22, and in an SAR-dependent increase in the rate of development of spontaneous mammary tumours and chemically-induced skin tumours. Repeated microwave exposure, followed by a "sub-carcinogenic" dose of carcinogen, resulted in an increased number of skin tumours. A study of 100 rats exposed for most of their lifetime at about 0.4 W/kg did *not* show any increased incidence of non-neoplastic lesions compared with control animals; longevity was very similar in both groups. However, the overall incidence of primary malignancy in the exposed group (18) was significantly greater than the control

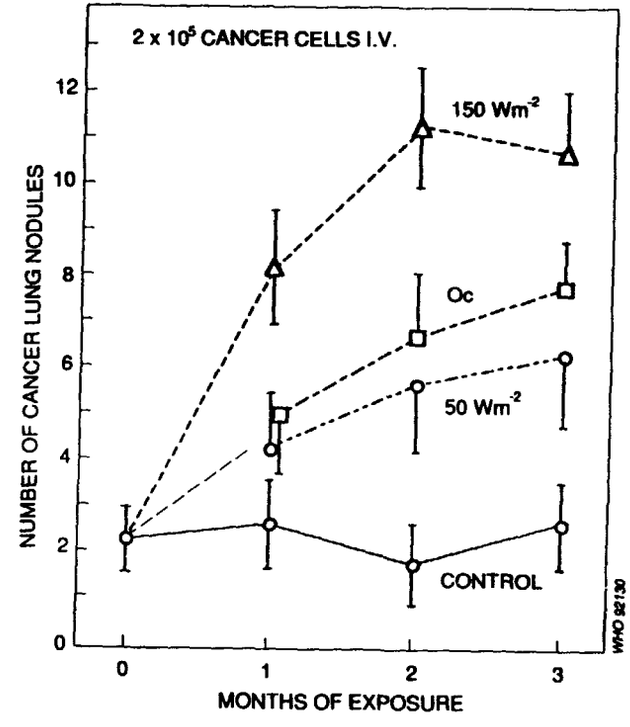

Fig. 22. Number of lung tumours (following intravenous injection of 2×10^5 viable sarcoma cells) in mice exposed to 2.45 GHz microwaves or non-specific stress (overcrowding; O_c). From Szmigielski et al. (1988).

value (5), but was reported to be similar to the spontaneous incidence given in the literature for the particular strain of rat. Under these circumstances, it is difficult to draw any firm conclusions.

Tumour weights were not significantly different in rats implanted with mammary adenocarcinoma tissue and either exposed 25 days later to 2 kHz magnetic fields of up to 2 mT for 1 h a day for 9 days or not exposed (Baumann et al., 1989). Handling and restraint stress in animals were identified as possible confounders for the detection of subtle magnetic field effects.

7.3.11 Summary and conclusions

Most of the biological effects of acute exposure to RF fields are consistent with responses to induced heating, resulting either in rises in tissue or body temperature of about 1 °C or more, or in responses for minimizing the total heat load. Most responses in different animal species, exposed under various environmental conditions, have been reported at SARs above about 1-2 W/kg.

These animal (particularly primate) data indicate the types of response that are likely to occur in humans subject to a sufficient heat load. However, direct quantitative extrapolation to humans is difficult, given species differences in responses, in general, and in thermoregulatory ability particularly.

The most sensitive animal responses to heat loads are thermoregulatory adjustments, such as reduced metabolic heat production and vasodilation, with thresholds ranging between about 0.05 and 5 W/kg, depending on environmental conditions. However, these reactions form part of the natural repertoire of thermoregulatory responses that serve to maintain normal body temperatures.

Transient effects seen in exposed animals that are consistent with responses to increases in body temperature of 1 °C or more (and/or SARs in excess of about 2 W/kg in primates and rats) include the reduced performance of learned tasks and increased plasma corticosteroid levels. Other heat-related effects include temporary haematopoietic and immune responses, possibly in conjunction with elevated corticosteroid levels. The most consistent effects observed are reduced levels of circulating lymphocytes and increased levels of

neutrophils, decreased natural killer cell function, and increased macrophage activation; an increase in the primary antibody response of B-lymphocytes has also been reported. Cardiovascular changes consonant with increased heat load, such as increased heart rate and cardiac output, have been observed, together with a reduction in the effects of drugs, such as barbiturates, the action of which can be altered by changes in circulation and clearance rates.

Most animal data indicate that implantation and the development of the embryo and fetus are unlikely to be affected by exposures that increase maternal body temperature by less than 1 °C. Above these temperatures, adverse effects, such as losses in implantation, growth retardation, and post-natal changes in behaviour, may occur, with more severe effects occurring at higher maternal temperatures.

Most animal data suggest that low RF exposure that does not raise body temperatures above the normal physiological range is not mutagenic; thus, such exposure will not result in somatic mutation or hereditary effects.

There is much less information describing the effects of long-term, low-level exposure. So far, it is not apparent that any long-term adverse effects can result from exposures below thermally significant levels. The animal data indicate that male fertility is unlikely to be affected by long-term exposure at levels insufficient to raise body and testis temperatures. Cataracts have not been induced in rabbits exposed at 100 W/m^2 for 6 months, or in primates exposed at 1.5 kW/m^2 for 3 months.

A study of 100 rats, exposed for most of their lifetime at about 0.4 W/kg, did not show an increased incidence of non-neoplastic lesions or total neoplasias compared with control animals; longevity was very similar in both groups. There were differences in the overall incidence of primary malignancies, but these could not necessarily be attributed to the RF exposure. The possibility that exposure to RF might influence the process of carcinogenesis is of particular concern. So far, there is no definite evidence that RF exposure does have an effect, but there is clearly a need for further studies to be carried out. Overwhelmingly, the experimental data indicate that RF fields are not mutagenic, and so they are unlikely to act as initiators of carcinogenesis. In a few studies, evidence has been sought of an enhancement of the effect of a known carcinogen.

The long-term exposure of mice at 2-8 W/kg resulted in an increase in the progression of spontaneous mammary tumours and of skin tumours in mice the skin of which was tested with a chemical carcinogen. Repeated RF exposure followed by a "sub-carcinogenic" dose of carcinogen resulted in an increased number of skin tumours; however, this study has been reported only briefly, and the authors noted the need for experimental confirmation.

In *in vitro* studies, enhanced cell transformation rates were reported after RF exposure at 4.4 W/kg (alone or combined with X-radiation) followed by treatment with a chemical promotor. The latter data have not always been consistent between studies. It is clear that studies relevant to carcinogenesis need replicating and extending further, to reduce uncertainties in this area.

A substantial body of data exists describing *in vitro* biological responses to amplitude-modulated RF radiation at SARs too low to involve any response to heating. Some studies have reported effects after exposure at SARs of less than 0.01 W/kg, occurring within modulation frequency "windows" (usually between 1 and 100 Hz) and sometimes within power density "windows".

Changes have been reported in the electroencephalograms of cats and rabbits, in calcium ion mobility in the brain tissue *in vitro* and *in vivo*, in lymphocyte cytotoxicity *in vitro*, and in the *in vitro* activity of an enzyme involved in cell growth and division. Some of these responses have been difficult to confirm, and their physiological or pathological consequences are not clear. However, any toxicological investigation should be based on tests carried out at appropriate levels of exposure. It is important that these studies be confirmed and extended to *in vivo* studies and that the health implications, if any, for exposed people are determined. Of particular importance, would be studies that link extremely low frequency, amplitude-modulated RF interactions at the cell surface with changes in DNA synthesis or transcription. It is worth noting that this interaction implies a "demodulation" of the RF signal at the cell membrane.

8. HUMAN RESPONSES

Epidemiology can be defined as the study of the occurrence of illness; its main goals are to evaluate hypotheses about the causation of illness and to relate disease occurrence to the characteristics of people and their environment. Epidemiological studies of human populations exposed to RF fields are few in number and are generally limited in scope. The principal groups studied have been people occupationally exposed in the military or in industry. Information about worker health status has generally come from medical records, questionnaires, and physical and laboratory examinations. Exposure data have come from personnel records, questionnaires, environmental measurements, and equipment-emission measurements. Determination of actual exposure to RF fields and to other risk factors for the same outcome is difficult in retrospective human studies.

Some studies of controlled exposures of volunteers have provided valuable information on responses to RF exposure. These studies include warming and pain thresholds for RF heating of the skin, RF hearing, and RF shocks and burns. Clinical studies of accidental overexposures provide information on acute-exposure responses.

8.1 Laboratory studies

8.1.1 Cutaneous perception

Exposure of the human body to RF fields can cause heating that is detectable by the temperature-sensitive receptors in the skin. Several investigators have determind experimentally the threshold intensities that cause sensations of perceptible warmth, pain, and delay in response to the stimulus in human subjects, as shown in Table 30.

Adair (1983a) noted that RF exposures to frequencies of 30 GHz and above would probably be similar to infrared in their perception threshold values. However, over much of the RF spectrum, current standards are set at levels that are below those that most would consider detectable by sensation. Thus, cutaneous perception may be an indicator of exposure only at RF frequencies of the order of several gigahertz or more, which have wavelengths that are small in comparison with the length of the exposed body, i.e., wavelengths

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